CLAIMS

1. A molecule having a nucleic acid sequence of from 48 to 1770 nucleotides and substantially identical to the corresponding nucleic acid sequence of SEQ ID NO. 2, wherein said sequence encodes a fragment of a peptide having SEQ ID NO. 1, or a homolog thereof, wherein said fragment or homolog enhances TNF-α transcription by interacting with a TNF- α promoter nucleotide sequence.

2. The molecule of claim 1, wherein said sequence encodes a LITAF DNA-binding domain for TNF-α promoter nucleotides CTCCC (–515 to –511).

3. The molecule of claim 1, wherein said fragment of peptide having SEQ ID NO. 1 comprises a LSQTWREPGAAGSPFHL peptide sequence.

4. The molecule of claim 1, wherein said TNF- α is human.

5. A vector comprising a molecule with said nucleic acid sequence of claim 1.

6. The vector of claim 5, wherein said vector is a viral vector.

7. The vector of claim 6, wherein said vector is selected from the group consisting of adenoviral vectors, adeno-associated virus (AAV) vectors, retroviral vectors, hybrid adenovirus-AAV vectors, and herpes-simplex virus (HSV) vectors.

8. A host cell containing a molecule having said nucleic acid sequence of claim 1.

9. An expression construct comprising a molecule with said nucleic acid sequence of claim 1.

10. The expression construct of claim 9, wherein said TNF- α is human.

11. The expression construct of claim 9, wherein said sequence encodes a LITAF DNA binding domain for TNF-α promoter nucleotides CTCCC (–515 to –511).

12. The expression construct of claim 9, wherein said fragment of peptide having SEQ ID NO. 1 comprises a LSQTWREPGAAGSPFHL peptide sequence.

13. A molecule having a nucleic acid sequence encoding a LSQTWREPGAAGSPFHL peptide sequence, or a homolog thereof.

14. A vector comprising said molecule of claim 13.

15. The vector of claim 14, wherein said vector is a viral vector.

16. The vector of claim 15, wherein said vector is selected from the group consisting of adenoviral vectors, adeno-associated virus (AAV) vectors, retroviral vectors, hybrid adenovirus-AAV vectors, and herpes-simplex virus (HSV) vectors.

17. A host cell containing said molecule of claim 13.

18. An expression construct comprising said molecule of claim 13.

19. A peptide fragment of a peptide having SEQ ID NO. 1, whereby said fragment enhances TNF-α transcription by interacting with a TNF- α promoter nucleotide sequence.

20. The peptide fragment of claim 19, wherein said TNF- α is human.

21. The peptide fragment of claim 19 comprising a SQTWREPGAAGSPFHL peptide sequence.

22. The peptide fragment of claim 19 comprising a naturally occurring allelic variant of a SQTWREPGAAGSPFHL peptide sequence.

23. A homolog of the peptide fragment of claim 19 comprising a variant of a SQTWREPGAAGSPFHL peptide sequence, wherein said variant contains a conservative amino acid substitution for a residue of said sequence, wherein said substitution does not adversely effect interaction of said fragment to said TNF- α promoter nucleotide sequence.

24. The peptide fragment of claim 21, wherein said fragment comprises an N-terminal truncation of said peptide having SEQ ID NO. 1.

25. The peptide fragment of claim 21, wherein said fragment comprises a C-terminal truncation of said peptide having SEQ ID NO. 1.

26. The peptide fragment of claim 21, wherein said fragment comprises an N-terminal truncation and a C-terminal truncation of said peptide having SEQ ID NO. 1.

27. A method for determining the inhibition of LITAF binding to a TNF-α promoter region by a compound comprising:

a) incubating a mixture of the following components:

i) a first molecule comprising a SQTWREPGAAGSPFHL peptide sequence, wherein said molecule is not full-length LITAF,

ii) a second molecule comprising said TNF-α promoter region, and

iii) said compound;

b) measuring the extent of binding of component i) to component ii) in the absence of component iii);

c) measuring the extent of binding of component i) to component ii) in the presence of component iii); and

d) determining the ratio of the binding measured in step c) to that measured in step b), a decrease of binding in step c) relative to step b) indicates that said compound inhibits the binding of LITAF to said TNF-α promoter region ion.

28. The method of claim 27, wherein said binding in steps b) and c) is expressed as a ratio of amount of component i) bound to component ii) to the amount of unbound component i).

29. The method of claim 27, wherein the incubation mixture of step a) is formed within a cell of a cell culture.

30. The method of claim 27, wherein the incubation mixture of step a) is formed in the absence of a cell.

31. The method of claim 27, wherein said TNF-α promoter region comprises nucleotides CTCCC (–515 to –511).

32. The method of claim 27, wherein said second molecule is fixed to a solid support.

33. The method of claim 27, wherein said binding of step b) results in the functional activation or repression of said TNF-α promoter region.

34. The method of claim 33, wherein said TNF-α promoter region is functionally linked to a second nucleic acid sequence encoding a reporter moiety and said binding of step b) results in the expression of said reporter moiety.

35. The method of claim 34, wherein said reporter moiety is luciferase.

36. The method of claim 34, wherein said reporter moiety is green fluorescence protein.

37. A method for determining the enhancement of LITAF binding to a TNF-α promoter region by a compound comprising:

a) incubating a mixture of the following components:

i) a first molecule comprising a SQTWREPGAAGSPFHL peptide sequence, wherein said molecule is not full-length LITAF,

ii) a second molecule comprising said TNF-α promoter region, and

iii) said compound;

b) measuring the extent of binding of component i) to component ii) in the absence of component iii);

c) measuring the extent of binding of component i) to component ii) in the presence of component iii); and

d) determining the ratio of step c) to step b), wherein an increase of binding in step c) relative to step b) indicates that said compound enhances the binding of LITAF to said TNF-α promoter region.

38. The method of claim 37, wherein said binding in steps b) and c) is expressed as a ratio of amount of component i) bound to component ii) to the amount of unbound component i).

39. The method of claim 37, wherein the incubation mixture of step a) is formed within a cell of a cell culture.

40. The method of claim 37, wherein the incubation mixture of step a) is formed in the absence of a cell.

41. The method of claim 37, wherein said TNF-α promoter region comprises nucleotides CTCCC (–515 to –511).

42. The method of claim 37, wherein said second molecule is fixed to a solid support.

43. The method of claim 37, wherein said binding results in the functional activation or repression of said TNF-α promoter region.

44. The method of claim 43, wherein said TNF-α promoter region is functionally linked to a second nucleic acid sequence encoding a reporter moiety and said binding of step b) results in the expression of said reporter moiety.

45. The method of claim 44, wherein said reporter moiety is luciferase.

46. The method of claim 44, wherein said reporter moiety is green fluorescence protein.

47. An antibody which binds to a LITAF peptide or fragment thereof, wherein said peptide or fragment comprises the SQTWREPGAAGSPFHL peptide sequence.

48. The antibody of claim 47, wherein said antibody is a monoclonal antibody.

49. A method of suppressing tumor cell growth in an animal comprising administering said vector of claim 5.

50. The method of claim 49, wherein said animal is a human.

51. The method of claim 49, wherein said tumor is a solid tumor.

52. The method of claim 51, wherein said tumor is of a cancer selected from the group consisting of non-small cell lung carcinoma, prostate carcinoma, renal carcinoma, colon carcinoma, ovarian carcinoma, pancreatic carcinoma and melanoma.

53. The method of claim 49, said method further comprising determining if said tumor cell is deficient in p53.